News from the Women’s Health Initiative: Reducing Total Fat Intake May Have Small Effect on Risk of Breast Cancer, No Effect on Risk of Colorectal Cancer, Heart Disease, or Stroke

Following an eating pattern lower in total fat did not significantly reduce the incidence of breast cancer, heart disease, or stroke, and did not reduce the risk of colorectal cancer in healthy postmenopausal women, according to the latest clinical trial results from the National Institutes of Health’s Women’s Health Initiative (WHI). The study was designed to evaluate a low-fat dietary pattern’s effect on the risk of cancer. However, investigators also evaluated the data to review the effect on cardiovascular disease. The results from the largest clinical trial ever of low-fat diet were reported in the *Journal of the American Medical Association*.

Among the 48,835 women who participated in the trial, there were no significant differences in the rates of colorectal cancer, heart disease, or stroke between the group who followed a low-fat dietary plan and the comparison group who followed their normal dietary patterns. Although the women in the study who reduced their total fat intake had a 9 percent lower risk of breast cancer than did women who made no dietary changes, the difference was not large enough to be statistically significant — meaning it could have been due to chance.

By the end of the first year, the low-fat diet group reduced average total fat intakes to 24 percent of calories from fat, but did not meet the study’s goal of 20 percent. At year six, the low-fat diet group was consuming 29 percent of calories from fat. The comparison group averaged 35 percent of calories from fat at year one and 37 percent at year six. Women in both groups started at 35-38 percent of calories from fat. The low fat diet group also increased their consumption of vegetables, fruits, and grains.

Women were aged 50-79 at trial enrollment in 1993-98 and were followed for an average of 8.1 years. The study diet focused on reducing total fat, and unlike diets used to reduce heart disease risk, did not differentiate between “good fats” found in fish, nuts, and vegetable oils, and “bad” fats like saturated fat and trans fat found in processed foods, meats, and some dairy products. The study design reflected a widely believed but untested theory that reduction of total fat would reduce risks of breast or colorectal cancers. For heart disease, it was anticipated that reduction in total fat would be accompanied by a reduction in saturated fats, which are known to contribute to heart disease risk.

“The results of this study do not change established recommendations on disease prevention. Women should continue to get regular mammograms and screenings for colorectal cancer, and work with their doctors to reduce their risks for heart disease including following a diet low in saturated fat, trans fat and cholesterol,” said National Heart, Lung, and Blood Institute Director Elizabeth G. Nabel, M.D.

The U.S. Dietary Guidelines for Americans recommend that adults keep total fat intake between 20 and 35 percent of calories, and saturated fats less than 10 percent of calories, with most fats coming from sources of polyunsaturated fats and monounsaturated fats, such as fish, nuts, and vegetable oils. For people with heart disease or at high risk for heart disease, targets for saturated fats may be further lowered.

“This study shows that just reducing total fat intake does not go far enough to have an impact on heart disease risk. While the participants’ overall change in LDL “bad” cholesterol was small, we saw trends towards greater reductions in cholesterol and heart disease risk in women eating less saturated and trans fat,” said Jacques Rossouw, M.D., WHI pro-

(Continued on page 2)
ject officer. The study also found that following a high-carbohydrate, low-fat eating pattern does not increase body weight, triglycerides or indicators of increased risk of diabetes such as blood glucose or insulin levels in women. The American Heart Association revised their 2000 guidelines this year. The updated 2006 dietary guidelines set a lower goal for saturated fat (less than 7% vs. less than 10% in the 2000 guidelines) and a goal for trans fatty acids of less than 1% of total calories.

“Study data indicate that women who started with the highest fat intake and who had greater changes in fat intake, show stronger evidence for reduction in their risk of breast cancer. Longer follow-up may be needed to show the effects of diet on cancer risk over time,” said Leslie G. Ford, M.D., National Cancer Institute.

Though the overall risk of colorectal cancer was unchanged in the dietary trial, secondary analyses suggested a possible benefit in women who were taking aspirin or combined hormone therapy (estrogen plus progestin); however, these findings could have occurred by chance. Polyps and adenomas (thought to be precursors of cancer) were reduced by 9%, suggesting that a benefit for colorectal cancer risk might emerge over time.

The WHI is the most comprehensive study to date of the causes and prevention of the major diseases affecting the health of older women. Over 15 years, the study’s findings on heart disease, breast and colorectal cancer, and osteoporosis have stimulated many changes in clinical practice. The WHI is also one of the largest studies of its kind ever undertaken in the United States and is considered a model for future studies of women’s health.
DHA DURING PREGNANCY—BETTER INFANT SLEEP AND PROBLEM-SOLVING

Carol J. Lammi-Keefe, Ph.D., R.D.

The importance of docosahexaenoic acid (DHA, 22:6n-3), a long chain n-3 polyunsaturated fatty acid, to infant development in the postnatal period is underlined by the inclusion of DHA and arachidonic acid in U.S. infant formulas since February 2002.

DHA status during pregnancy impacts the central nervous system (CNS), learning and cognition from birth to toddlerhood (1, 2). The benefit of maternal DHA status during pregnancy to infant behavioral states immediately after birth has been demonstrated previously (1). Cheruku et al assessed sleep patterning in the first 48 hours after birth. That is, for instance, how much time the infant spent in quiet versus active sleep, which is similar to REM sleep in adults; and how much total time each day was spent in a sleep state versus a wakeful state. The significance of the finding is that sleep patterning represents maturity of the CNS and thus supports the notion maternal DHA status during pregnancy can impact development of fetal CNS development (1). Others have provided evidence for the influence of DHA during pregnancy on habituation in the first year and free-play attention and distractibility during toddlerhood (2). Habituation is a fairly basic tool for assessing the speed of information processing and recognition memory in infants. Both habituation and distractibility relate to how quickly an infant or toddler processes information and this relates to infant cognition. With DHA supplementation both during pregnancy and during the first few months of breastfeeding higher I.Q. was documented in a group of Norwegian children at four years of age (3). This report provides evidence for an effect into the preschool years (3).

We undertook an intervention study to determine the effect of supplemental DHA during pregnancy (average 214 mg DHA/day) versus a placebo on infant functional outcomes immediately after birth and at 9 months. The following describes in brief the methods and the results for our study.

Pregnant women were recruited prior to 22 weeks of pregnancy. Fourteen women were in the experimental group and 15 in the control group. At 24 weeks of pregnancy the women were randomly assigned to consume either a functional food with DHA or a placebo. The food was consumed until delivery of their babies. The functional food was a cereal based bar which the women consumed by itself or in combination with their usual foods. At birth, infant sleep patterns were assessed for 48 hours using the Motility Monitoring System (MMS) (4). The MMS consisted of a mattress pad that fit under the baby’s blanket in the hospital crib. The mattress was attached to an amplifier and a data logger contained in a brief case hung on the crib stand. The instrumentation was battery operated, allowing the crib cart to be moved wherever necessary. There were absolutely no attachments made to the baby, i.e., no electrodes. Because the mattress was pressure sensitive, movements related to both body movements and respiration of the infant were recorded. The patterns that were generated were compared to prototypes characterizing the sleep as active sleep, quiet sleep, wakefulness, and arousals. The percent of time in each state was calculated.

With respect to the infant sleep patterning, there have been no other studies to determine the effect of intervention with DHA during pregnancy on this most immediate postnatal state. In this randomized, double-blinded and placebo-controlled trial on the first day after birth there were differences between groups in arousals, in quiet sleep and in active sleep. The group of infants born to women who consumed the supplemental DHA had fewer arousals than the placebo group (6). These data speak to the sensitivity of the functional assessment methodology, as differences in maternal DHA levels were not apparent. The data support the notion that a moderate increase in DHA during pregnancy can have functional consequences for the developing infant immediately after birth. While arousals during sleep are a crucial and protective response in infants, repeated arousals during sleep may result in sleep fragmentation that can lead to decreased arousal response to respiration and therefore detrimental outcome for the infant. Small increases in DHA during normal pregnancy appear to impact neurological development of the infant that expresses itself immediately after birth.

In this cohort of pregnant women and infants the problem-solving ability of the infants at 9 months was assessed using the Infant Planning Test (5), a two-step problem solving trial. The infants were presented with a problem-solving trial consisting of a toy covered with a cloth sitting on a second cloth. To ‘solve’ the problem the infant needs to accomplish...
pull the cloth to get access to the covering cloth and toy, lift the covering cloth to uncover the toy and grab the toy to play with it. This methodology has been well described (5).

The infants born to the women who consumed the DHA functional food during pregnancy performed in a superior manner compared to the infants born to the women who consumed the placebo (7). The infants of the women who consumed the supplemental DHA either completed or showed more intentions to complete more steps in the trial, such as grabbing the cloth to pull the object within reach, uncovering the toy and grasping the toy to play with it. The DHA benefit may affect the attention processes and psychomotor systems, but this will need further exploration.

In this study the gestational length was affected. Gestational length was significantly increased by the consumption of the supplemental DHA, an effect that has previously been documented (8). This benefit of DHA is especially important for women at increased risk for preterm delivery.

In summary, pregnant women should be encouraged to increase intake of DHA. Most recent estimates of dietary DHA intake by this population puts the intake at approximately 50 to 100 mg/day, less than the current recommendation of 300 mg/day for pregnant and lactating women (8). Good sources of DHA include coldwater marine fish of non-predator species, fish oils, high-DHA eggs and DHA from single-cell, algal source. Education about the benefits of DHA to infant neurodevelopment needs to reach healthcare providers who are advising women in preparation for successful pregnancy.

References:
Introduction
Mercury (Hg) is a public health concern due to its widespread persistence in the environment and known toxic effects\(^1\). People are exposed to methylmercury (MeHg) mainly through consumption of predatory fish species, and inorganic mercury (I-Hg) through the release of mercury vapor from amalgam dental fillings\(^2\). Increased maternal mercury levels during pregnancy result in the accumulation of both MeHg and I-Hg in the fetal brain, both of which can cause neurotoxic effects\(^3\). Prenatal MeHg exposure can result in developmental delay and cerebral palsy\(^3\). MeHg poisoning is characterized by loss of sensation to the extremities and mouth, ataxia, dysarthria, decreased vision, and loss of hearing\(^3\). Severe poisoning can cause blindness, coma, and death\(^3\).

In infants, the risk of Hg exposure occurs primarily from the consumption of Hg-contaminated breast milk. Infant Hg intake is influenced by maternal dietary habits and the chemical form of Hg\(^1\). MeHg interferes with cell proliferation and migration of neurons in the central nervous system (CNS)\(^3\). Cortical migration is not fully complete until 5 months of age, and neurons continue to proliferate during the first year of life\(^4\). Therefore, the postnatal brain is at risk for toxic exposures to harmful substances such as Hg.

Two outbreaks of MeHg toxicity have been well documented\(^5\). In Iraq, wheat laced with MeHg-containing fungicide was used to make bread. In Japan, the consumption of fish contaminated with MeHg by industrial pollutants in Minamata Bay caused infants to be born with CNS defects. These unfortunate instances led to the recommendation for restricting consumption of MeHg-containing...
To determine Hg intake Bjornberg et al\textsuperscript{2} distributed a questionnaire concerning fish intake for the previous 6 months. Each mother completed the questionnaire at time of delivery and 13 weeks postpartum. At 13 weeks information about breast feeding and the use of infant formula was also included. The mean fish intake during breastfeeding was 2.4 times per month, compared to 2.0 during pregnancy. The questionnaire did not ask if predatory type fish were eaten. The study found no significant correlations between reported fish intake and maternal or infant blood MeHg. Neurological effects were not reported on, and the exact maternal Hg consumption was not quantified. It is possible that maternal MeHg consumption was inadequate to result in postnatal CNS complications.

Sakamoto et al\textsuperscript{5} also studied MeHg levels in infants who were reared on breast milk to evaluate the risks of exposure between fetal and breast feeding periods. They used Hg concentrations in red blood cells (RBC) and plasma as markers. RBC-Hg was used as a biomarker for the body’s level of MeHg. Plasma-Hg was used to determine I-Hg exposure. Seven women and their infants were studied. The maternal RBC to plasma ration of Hg concentration was consistent with ratios in populations with increased fis intake\textsuperscript{6}. In all samples, RBC-Hg in the umbilical cord was higher than maternal blood at delivery. This supports previous study results that showed a correlation between maternal blood and umbilical cord blood levels of MeHg at delivery. Umbilical plasma-Hg was similar to plasma-Hg in the mothers at delivery. All the infants showed a significant decline in RBC-Hg concentration throughout breastfeeding. At 3 months, maternal RBC-Hg concentrations were significantly higher than their infants. This result coincides with the same pattern seen in the Bjornberg et al study. Breast milk Hg was assessed at 3 months after delivery. Significant correlations between maternal RBC-Hg and milk-Hg were seen, as well as with plasma-Hg and milk-Hg. Sakamoto et al found the contribution of breast milk to MeHg concentrations in infants appeared limited. They proposed the rapid increase in body weight and the limited Hg transfer from breast milk might have caused the dilution in RBC-Hg levels during the three months after delivery. This supports the thesis that infant MeHg exposure during breastfeeding does not result in significant neurological deficits even in populations with a high fish intake.

Jenson et al\textsuperscript{8} examined the result of Hg exposure during breast feeding on neurobehavioral performance of the child at age 7 years in the Faroese Islands. One thousand twenty-two singleton births at three Faroese hospitals were followed. Umbilical cord blood was obtained at delivery. At delivery, midwives interviewed mothers about their nutrition habits concerning the frequency of dinners with fish or pilot whale. After delivery, nurses visited the family at home several times during the infant’s first year of life. The last visit occurred around 12 months of age. Milestone developments, total number of months breastfeeding, and a hair sample were collected from the child for Hg analysis. Unfortunately only 52% or 572 infants had the final exam completed. Maternal Hg-concentration in hair at time of delivery was slightly higher in MeHg than infant hair at 12 months. The authors believed the higher intake of whale in the smaller villages was the reason that children visited in the villages had a higher hair MeHg level than children who...
lived in the capital city.

A total of 917 children completed the neuropsychological tests at age 7. History of breastfeeding was provided by 905 mothers, and 846 reported the total length of breastfeeding. All but 70 of the children were breastfed and 61% were exclusively breastfed for 0-4 months. Only 5% were exclusively breastfed for greater than 6 months. They found children who were breastfed longer had a significantly higher hair mercury concentration at age 1 year. Children who were breastfed longer performed slightly better on most neuropsychological tests before confounder adjustment. After adjustment, the positive effect of breastfeeding was reduced, although breastfed children still performed slightly better on most tests. The study then looked at specific tests they thought would be most affected by Hg exposure. These were hand-eye coordination, continuous performance test, an intelligence scale for children, and a naming test. They tested looked at children with higher levels of umbilical and hair Hg and then correlated it with length of breastfeeding. Jensen et al found only a negligible difference in breastfed infants compared to infants who were not breastfed for less time. Higher hair mercury at age 1 was associated with better test scores, but the regression coefficients for hair Hg at age 1 were clearly reduced after inclusion of breastfeeding. In this cohort of children with a relatively high prenatal mercury exposure and potential exposure through breast milk, breastfeeding was not associated with any deficit in neuropsychological performance at age 7. Breastfeeding did not appear as beneficial as reported by other investigators in non Hg exposed populations. In regard to neurotoxic risks, this study concluded women can be advised to breastfeed their children even in a community with relatively high exposure to Hg. This also supports the thesis of this paper.

In Iraq, in 1972, an epidemic of MeHg poisoning in farmers and their families occurred3. A total of 6530 cases were admitted to the hospital and 459 hospital deaths reported. Exposure to MeHg occurred from home-made bread prepared from wheat flour treated with a methyl-mercurial fungicide. It was determined that MeHg was the only harmful compound in the wheat and barley samples, and no other foods regularly consumed were found to have toxic substances. The hospital admission frequency of the 0 to 1 age group was the lowest of all age groups, making up less than 1% of the cases. Approximately half of the children of this age group were born before the onset of the poisoning occurred. Therefore, their exposure to MeHg occurred primarily through breast milk.

In Bakir et al3 study samples of whole blood were analyzed. The blood contained mostly MeHg located in the RBCs. Compared to whole blood, plasma and breast milk concentration of T-Hg was much lower and the proportion of I-Hg was higher. The total amounts of Hg in plasma and breast milk correlated closely with the corresponding amounts in blood. As the mean period of ingestion increased, the concentration of Hg in the blood increased, resulting in more symptoms of Hg poisoning.

Samples of maternal blood and milk were collected from 20 lactating women. The concentration of MeHg in milk was proportional to the concentration of MeHg in maternal blood. The Hg concentration in milk averaged 3% of the mean concentration in blood.

Paired maternal and infant blood samples were collected from 11 cases where the infant was born before the epidemic and thus exposed only to MeHg in human milk. The infants ranged from 9 to 18 months old. The MeHg concentration in the blood of each infant was either equal to or lower than that of its mother. No signs of Hg poisoning were reported in the infants at the time of the study. Bakir et al3 found that compared to infants who had been exposed in utero, breast fed infants were at a decreased risk for physical signs of MeHg poisoning. In summary they found that MeHg is transferred into milk at a concentration equal to 3 percent of the concentration in blood. They hypothesized this could lead to a hazardous concentration in a lactating infant if the MeHg concentration of the mother is high.

Amin-Zaki et al9 completed a 5-year longitudinal study after the Iraq epidemic and found mothers with the most severe symptoms of Hg poisoning had an average blood Hg concentration significantly higher than either the milder or asymptomatic groups. Analytical data indicated the predominant route of exposure for the infant was through breast milk in which approximately 60% of total Hg was determined to be MeHg. Abnormal neurological signs in 8 of 22 infants occurred at the first examination, and 17 of 22 at the second examination. Delayed motor development became evident at the second and third examinations. The frequency of pathological reflexes and delayed motor developmental milestones was so high as to be considered significant even in the absence of a controlled study. This refutes...
the thesis that Hg exposure from human milk does not result in significant neurological deficits.

Sundberg et al\textsuperscript{3} studied the risk of Hg accumulation and toxicity in mice exposed to MeHg or I-Hg through milk. One limit to this study is that they were unable to speciate Hg in the milk. Therefore, it is uncertain whether the milk truly contained MeHg. It was determined the absorption process in the GI-tract is not the same for I-Hg and MeHg. The result of this study supports the recommendation that lactating women should avoid consumption of certain species of freshwater fish in order to prevent contamination of breast milk with MeHg.

Discussion

The benefits of breastfeeding have been well documented\textsuperscript{2}. These benefits may outweigh the possible adverse effects of Hg in breast milk. However, if mothers are exposed to high levels of Hg during lactation, caution is recommended concerning breast feeding. Significant correlations between maternal plasma-Hg and milk-Hg and between maternal RBC-Hg and MeHg were observed in the Sakamoto et al\textsuperscript{5} study. This suggests maternal MeHg levels affect milk Hg levels. After consumption of MeHg contaminated bread in Iraq, Hg levels in blood and breast milk were correlated with milk levels being about 3% of the blood levels. Therefore, one might see an increased amount of Hg transferred to the infant if the mother’s exposure is high.

It would be beneficial if the studies reviewed controlled for actual Hg ingestion by the mother to determine how blood levels are affected. In the studies reviewed that supported the thesis, small sample size was a limitation. Due to ethical concerns, a randomized control study in humans is not possible.

The WHO considers concentration ranges of 1.4 to 1.7 ng Hg/g as “normal” in breast milk\textsuperscript{10}. These studies showed that Hg was not elevated above the “normal” limit. Only the Iraqi women had T-Hg milk concentrations higher than “normal.” This may explain why breastfeeding appeared to pose a minimal threat to the postnatal brain.

Unfortunately many women avoid eating all fish for fear of ingesting harmful levels of MeHg. Fish contains many beneficial components that may also cause a person to be healthier, such as omega-3 fatty acids and high quality proteins, and being low in saturated fat\textsuperscript{11}. Though maternal Hg can be transferred to infants through breast milk, exposure through the placenta during their intrauterine life appears to be a greater risk for neurotoxic effects. Bjornberg et al\textsuperscript{2} and Sakamoto et al\textsuperscript{5} showed Hg-concentration in blood declines in the breastfed infant as the infant gets older. However in Amin-Aakin et al’s\textsuperscript{9} longitudinal study of children exposed to MeHg in Iraq and Sundberg et al\textsuperscript{11}’s study in mice it appears that increased maternal MeHg intake can result in the accumulation of Hg in the offspring further from the time of exposure to Hg. Without knowledge of true maternal MeHg intake it is difficult to assess an upper limit for Hg consumption during breastfeeding. Even if an upper limit was known, the content of MeHg in predatory fish varies for different physical locations. Guidelines would be different for different locations. Thereby, a world-wide standard for fish intake is impossible to determine. It would be best to choose the strictest level to avoid neurotoxic symptoms in breastfed infants.

References

2. Bjornberg K et al. Transport of Methylmercury and Inorganic Mercury to the Fetus and Breast-fed Infant. \textit{Environmental Health Perspectives} 2005; 113(10), 1381-1385.
Upcoming Conferences—Compiled by Alison Starr

Taken from NIH – Division of Nutrition Research Coordination: http://www.cnrc.nih.gov/dnrc/calendar.htm

September 2006

- 7-9 - The University of Kansas 8th Annual Conference on the Prevention and Treatment of Overweight and Obese Individuals. Hyatt Regency, Crown Center, Kansas City, MO. Contact Kim Johnson; (785) 864 0797; kim@ku.edu; http://www.ebl.ku.edu/sindex.htm

- 10-12 - Robert Wood Johnson Foundation Forum for State Legislators and State Policymakers: Healthy Lifestyle Behaviors: Childhood and Beyond. Location TBD, Contact Robby Morton; 859.244.8254; rmorton@csg.org; http://www.healthystates.csg.org/Events+and+Conferences/RWJF+Forum.htm

- 12-14 - CDC's 2006 National Health Promotion Conference. Innovations in Health Promotion: New Avenues for Collaboration. Hilton Atlanta, Atlanta, GA. Contact Conference Manager; (770) 488-6509; chronicconf@cdc.gov; http://www.cdc.gov/cochp/conference/


- 16-19 - American Dietetic Association Food and Nutrition Conference and Exposition. Hawaii Convention Center, Honolulu, HI. Contact ADA, 216 West Jackson Boulevard, Chicago, IL 60606-6995; fax 312 899-0008; mtgsinfo@eatright.org; http://www.eatright.org/cps/rde/xchg/ada/hx.xsl/7539_ENU_HTML.htm


- 17-20 - World Grains Summit: Foods and Beverages. Moscone Convention Center, San Francisco, CA; Call 651-454-7250; aacc@scisoc.org; http://www.wgsummit.org

- 17-21 - IUFOst 13th World Congress of Food Science and Technology. Nantes, France; iufost@nantes.inra.fr; http://www.iufost.org

- 19-20 - 30th US National Nutrient Databank Conference. Honolulu, HI. Contact Jean Pennington; ip157d@nih.gov or Phyllis Stumbo; Phyllis-stumbo@uiowa.edu; http://http://www.eurofir.net/public.asp?id=2252

- 19-21 - Tufts University Friedman School Symposium: “Nutrition is not a discipline, it is an agenda”. Boston, MA. Contact Mark Krumm; 617-636-3718; mark.krumm@tufts.edu; http://nutrition.tufts.edu/conferences/symposium/


Ask The Experts:

An informal survey of members was conducted to determine current practice with regard to DHA supplementation. Here are the results of almost 60 respondents.

Do you think pregnant women should be supplemented with DHA if their dietary intake is low?

- Almost 64% of respondents reported yes, 4 percent said no, and 32% were not sure.

Do you think that women with cardiovascular disease or who are at high risk for CVD should take DHA supplements?

- Fifty-two percent of respondents said yes, 7% said no and 41 percent said they were not sure.

Should breastfeeding women be encouraged to take DHA supplements if dietary intake is low?

- Sixty percent of WHRN members who responded said yes, 5% said no and 35% were unsure.
Upcoming Conferences-Continued

(Continued from page 9)

- 28 - Current Concepts in Nutrition and Aging. Pyle Center, Madison, WI. Contact University of Wisconsin; 1-888-391-4255; elaine.barrett@uwex.edu; http://www.uwex.edu/ces/flp/conference/

- 28-29 - Mayo Clinic Nutrition in Health and Disease. Marriott Minneapolis City Center, Minneapolis, MN; (800)-323-2688; cme@mayo.edu; http://www.mayo.edu/cme/internal-medicine.html

October 2006

- 1-4 - 4th European Federation of Lipids Congress. Madrid, Spain; http://www.eurofedlipid.org/meetings/madrid/index.htm

- 5-8 - American College of Nutrition's 47th Annual Meeting- Reno-Hilton Hotel, Reno, NV; 727-446-6086; office@amcollnutr.org; http://www.amcollnutr.org

- 7-11 - Tenth Annual Conference of the Community Food Security Coalition in Conjunction with Food Secure Canada. Vancouver, Canada; http://www.foodsecurity.org

- 8-10 - International Food and Nutrition Conference. Kellogg Conference Center, Tuskegee, AL. Contact Adelia Bovell-Benjamin; 334-727-8717; IFNC2006@gmail.com

- 23-26 - Treating the Dieting Casualty. Gateway Crossing Community Center, Hagerstown, MD. Contact Nutrition/Wellness Services; 240-313-3300; CWeaver@dhmh.state.md.us


- 26-27 - International Probiotics Symposium. Montreal, Canada. Contact Yvan Grégoire; CournoyerE@AGR.GC.CA; http://www.probiomtl.org/2006/index.htm


- 26-29 - World Congress on Controversies in Obesity, Diabetes and Hypertension (CODHy). Hotel Estrel, Berlin, Germany. Contact Michal Pink; 33-170-367-820; fax 33-170-367-821; codhy@codhy.com; http://www.codhy.com

November 2006

- 3-4 - Food Chains: Provisioning, Technology, and Science. Contact Carol Lockman, Hagley Museum and Library, Wilmington, DE; 302 658-2400, ext 243; fax 302 655-3188; clockman@hagley.org

- 4-8 - American Public Health Association 133rd Annual Meeting and Exposition. Boston, MA; Call 202 777-2742; http://www.apha.org/meetings


- 16-17 - Income Volatility and Implications for Food Assistance Programs II. Washington, DC; http://www.npc.umich.edu/news/events/USDA/
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Are you interested in our efforts to support breastfeeding?

If supporting breastfeeding is important to you, join our WHRN Breastfeeding Task Force. Our next meeting is Tuesday, September 19 2:30pm-4:30pm, Hilton Hawaiian Village, Sea Pearl I. If you can’t make it to Hawaii, keep an eye out for upcoming conference calls. Calls are announced on the listserv (WHRN_list-subscribe@yahooogroups.com).


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DPG ACTIVITIES AT FNCE 2006—Laura Couillard, RD, FNCE Coordinator 2006

Saturday, September 16 Sheraton Waikiki, Kona Room:
• 7:00am-3:00pm, Executive Committee meeting
• Afternoon: Executive committee with Committees chairs and Coordinators
• 12:30: Membership lecture (open to all members and guests), RSVP to whrrnsvp@yahoo.com. Hot Topics in Women's Health — The Role of Soy Products, by Mark Messina, PhD, sponsored by the United Soybean Board.

Sunday, September 17th
• 1:30pm-2:30pm, Hawaii Convention Center, Room 316 ABC; “Rethinking Perinatal Vitamin D Intake: Is the Current Recommendation from the DRI’s Enough?” with Bruce Hollis, PhD
• 5:30 - 7:30pm, Hilton Hawaiian Village, South Pacific I: Membership Reception sponsored by Martek. The reception will include an educational presentation by Carol J. Lammi-Keefe, Ph.D., R.D. entitled Smarter, Better Sleeping Babies—Can What the Mother Consumes During Her Pregnancy Make a Difference?

Monday, September 18
• 10:30am – 1:00pm, Hawaii Convention Center, 3rd Floor: DPG Showcase

Tuesday, September 19
• 2:30pm-4:30pm, Hilton Hawaiian Village, Sea Pearl I: WHRN Breast Feeding Task Force meeting

We will be helping to sponsor the Mother's Room again this year. Volunteers are needed for the room. Please contact Cathy Fagen at 562-595-7930 or cfagen@memorialcare.org or Krista Neal at 512-569-6400 or kristakaye@hotmail.com for information.